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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
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EXAMINER	
WILSON, MICHAEL C	
ART UNIT	**PAPER NUMBER**
1632	16

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Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No. 09/557,907	Applicant(s) HORTON ET AL.	
	Examiner Michael Wilson	Art Unit 1632	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *19 March 2002* .

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1,3-7,9-22,30-35,38-50,66,67,69-74,77-81 and 83-86* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1,3-7,9,10,15-18,30-35,38-50,66,67,69-74,77-81 and 83-86* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

11)☐ The proposed drawing correction filed on _____ is: a)☐ approved b)☐ disapproved by the Examiner.

If approved, corrected drawings are required in reply to this Office action.

12)☐ The oath or declaration is objected to by the Examiner.

Priority under 35 U.S.C. §§ 119 and 120

13)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____ .

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

14)☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. § 119(e) (to a provisional application).

a) ☐ The translation of the foreign language provisional application has been received.

15)☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. §§ 120 and/or 121.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO-1449) Paper No(s) 6 .
4) ☐ Interview Summary (PTO-413) Paper No(s). _____ .
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☒ Other: *detailed action* .

DETAILED ACTION

The Art Unit location of your application in the USPTO has changed. To aid in correlating any papers for this application, all further correspondence regarding this application should be directed to Art Unit 1632.

Claims 2, 8, 23-29, 36, 37, 51-65, 68, 75, 76, 82 and 87-103 have been canceled. Claims 1, 3-7, 9, 10, 15-18, 30-35, 38-50, 66, 67, 69-74, 77-81 and 83-86 are being examined as they relate to administering DNA encoding IFN-α. The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office action. Applicant's arguments filed 3-19-02, paper number 14, have been fully considered but they are not persuasive.

Election/Restriction

This application contains claims 11-14 and 19-22 drawn to an invention nonelected with traverse in Paper No. 12. A complete reply to the final rejection must include cancellation of nonelected claims or other appropriate action (37 CFR 1.144) See MPEP § 821.01.

Claim Rejections - 35 USC § 112

1. Claims 1, 3-7, 9, 10, 15-18, 30-35, 38-50, 66, 67, 69-74, 77-81 and 83-86 remain rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for administering a plasmid encoding IFN-α operably linked to a promoter intramuscularly to a mouse having a tumor such that a decrease in tumor volume, a decrease in tumor metastases and an increase in survival occur, or intraparitoneally such that an increase in survival occurs, does not reasonably provide enablement for any non-infectious, non-integrating DNA other than plasmid,

active fragments of IFN-α, administering the composition to smooth muscle or myocardial tissue, obtaining cell-, tissue- or tumor-specific expression of IFN-α, administering a vector encoding IFN-α as well as another cytokine or treating any symptom of cancer as broadly claimed. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention commensurate in scope with these claims.

The state of the art with respect to IFN-α and cancer therapy at the time of filing can be well established in review articles published by Henderson (1992, Trends Pharm. Sci., Vol. 13, pages 145-152), Sedlacek (Critical Reviews in Oncogenesis, Vol. 5(6): 555-587, 1994), Dalgleish (Cancer Surveys, Vol. 26: 289-320, 1995), Belldegrun (1993, J. NCI, Vol. 85, pages 207-216), Santodonato (Jan. 1, 1996, Human Gene Therapy, Vol. 7, pages 1-10), Kaido (1995, Int. J. Cancer, Vol. 60, pages 221-229), Zhang (April 1996, PNAS, Vol. 93, pages 4513-4518), Zhang (1996, Cancer Gene Therapy, Vol., 3, pages 31-38) and Pestka (WO 97/00085, Jan. 3, 1997).

Henderson reviewed the properties of IFN-α (page 146, Table 1). Sedlacek provided a review of treatment of tumors in general and detailed descriptions of immune pathways known to be generated by various cytokines (see pages 568-575). Sedlacek emphasized the difficulty in treating tumors due to their resistance to immune recognition (see abstract and page 571, column 1, first full paragraph). Sedlacek specifically indicated that the combination of methodologies required to generate a specific immune response against tumors has not yet been discovered (page 575). Dalgleish reviewed the importance of IFN-α in generating tumor specific immune

responses (page 299, line 6), and indicated that "different cytokines can induce different anti-tumor immune responses offers boundless opportunities for combination as well as the potential for synergy" (pages 298-230). Pestka taught using tumor cells transfected with a plasmid encoding IFN-α to treat cancer (page 44, line 15).

Specifically, Belldegrun taught renal carcinoma cells transfected with a plasmid encoding IFN-α had decreased tumorigenicity in mice as compared to non-transfected cells. Santodonato taught administering Friend erythroleukemia cells (FLC) comprising a plasmid encoding IFN-α to pre-established FLC tumors caused tumor inhibition (page 3, col. 1, 11 lines from the bottom). Kaido taught that melanoma cells transfected with a plasmid encoding IFN-α had decreased tumorigenicity in mice as compared to non-transfected cells. Zhang taught that a number of tumors transfected with a retroviral vector encoding IFN-con1 had decreased tumorigenicity in mice as compared to non-transfected cells. IFN-con1 has significant homology to IFN-α and shares the most frequent amino acids in eight of the different IFNs (page 31, 12 lines from the bottom). Thus, it is clear from the prior art that the potential for treating various tumors using DNA encoding IFN-α existed.

At the time of filing, the combination of vector, promoter, protein and route of administration required to obtain a particular effect and to target desired tissues *in vivo* continues to be unpredictable and inefficient as supported by numerous teachings available in the art. For example, Miller (1995, FASEB J., Vol. 9, pages 190-199) review the types of vectors available for *in vivo* gene therapy, and conclude that "for the long-term success as well as the widespread

applicability of human gene therapy, there will have to be advances...targeting strategies outlined in this review, which are currently only at the experimental level, will have to be translated into components of safe and highly efficient delivery systems" (page 198, column 1). Deonarain (1998, Expert Opin. Ther. Pat., Vol. 8, pages 53-69) indicate that one of the biggest problems hampering successful gene therapy is the "ability to target a gene to a significant population of cells and express it at adequate levels for a long enough period of time" (page 53, first paragraph). Deonarain reviews new techniques under experimentation in the art which show promise but states that such techniques are even less efficient than viral gene delivery (see page 65, first paragraph under Conclusion section). Verma (Sept. 1997, Nature, Vol. 389, pages 239-242) reviews vectors known in the art for use in gene therapy and discusses problems associated with each type of vector. The teachings of Verma indicate the combination of vector, protein, promoter and route of administration required to obtain the desired effect and to target desired cells is unpredictable (see entire article; page 240, sentence bridging columns 2 and 3). Crystal (1995, Science, Vol. 270, page 404-410) also reviews various vectors known in the art and indicates that "among the design hurdles for all vectors are the need to increase the efficiency of gene transfer, to increase target specificity and to enable the transferred gene to be regulated" (page 409). Roth (1997, J. National Cancer Inst., Vol. 89, pages 21-39) taught non-viral vectors did not target cells of interest and provided low efficiency (page 26, col. 2).

Applicants argue Henderson, Sedlacek and Miller do not reflect the state of the art at the time of filing because they were published well before the filing date of the instant application.

Applicants argument is not persuasive because the information discussed in Henderson, Sedlacek and Miller had not been disproved at the time of filing. Therefore, the information provided by Henderson, Sedlacek and Miller reflects what one of skill in the art would have known about IFN-α and gene therapy at the time of publication as well as at the of filing.

Applicants argue Miller does not correlate to the instant invention because it discusses "replacement" therapy. Applicants argument is not persuasive because Miller discusses the targeting of vectors at the cell surface level and genetic level. The teachings of Miller are not limited to "replacement" therapy. In addition, Miller discusses non-"replacement" therapy (pg 193, col. 2, line 4, carcinoma; pg 195, col. 2, B-cell lymphoma; 196, col. 1, cancer). Finally, applicants invention relates to "replacement" therapy as taught by Miller because administering a plasmid encoding IFN-α could be used to increase IFN-α levels in a patient having low IFN-α levels.

Applicants argue Miller does not correlate to the instant invention because the statement cited on pg 198 is not based on fact. Applicants argument is not persuasive because the citation is a conclusory statement by Miller, one of skill in the art, based on the data from research involving retroviral vectors (pg 190), adenoviral vectors (pg 192), liposomes (pg 193), plasmids (pg 194, Fig. 3), et al. Applicants have not provided any evidence to the contrary that the conclusory statement by Miller is incorrect.

Applicants argue the conclusory statements by Deonarain and Verma are not supported by data. Applicants argument is not persuasive because the statements are based on data from

research involving numerous vectors (see entire articles). A statement that the art "will become as routine as practice as heart transplants" (Verma), that gene therapy has potential (Crystal, pg 409) or that plasmids is a promising area of vector development (Roth) indicates that the state of the art was that gene therapy was not routine and that the potential of gene therapy, specifically plasmids, had not been realized. Deonarain, Verma and Roth do not teach the administration of non-infectious, non-integrating vectors encoding protein targeted the desired tissue or produced any therapeutic effect. In fact, Roth teaches administering plasmid causes non-specific uptake (pg 26, col. 2, last sentence of first para.).

Applicants argue Deonarain does not relate to the instant invention because it relates to ligand-targeted receptor mediated endocytosis which is distinct from the claimed invention. Applicants argument is not persuasive because the claims do not exclude using ligand-targeted receptor mediated endocytosis to target the cells of interest. Moreover, the concept of targeting in gene therapy discussed by Deonarain directly correlates to the claimed invention because the vector encoding IFN-α as claimed must target the correct cell to produce a desired effect.

Applicants argue Crystal does not relate to the claimed invention because it discusses viral vectors. Applicants argument is not persuasive because the claims encompass non-infectious, non-integrating viral vectors. Applicants argue Crystal

The references cited above taken as a whole reflect the knowledge of one of skill in the art at the time the invention was made regarding the function of IFN-α, the ability to obtain a

therapeutic effect using non-infectious, non-integrating vectors for gene therapy, and the need to target tissues using such vectors.

The specification teaches making plasmids VR4101 and 4111 which encode mouse IFN-α and VR4102 and VR4112 which encode human IFN-α (page 92, line 24 - page 93, line 9). The plasmids were administered to mice having established melanoma, glioma or epidermoid carcinoma intramuscularly (page 100, line 33 - page 101, line 16; page 101, line 29). IFN-α was expressed to detectable levels in the serum in mice injected five times intramuscularly with 100 µl of VR4111 (page 103, line 13). Fig. 3 and 4 show mice having melanoma, glioma or epidermoid carcinoma receiving plasmid encoding mouse IFN-α intramuscularly experienced a decrease in tumor volume and an increase in survival as compared to control mice (page 104, line 11-22). Fig. 6 and 7 show mice having melanoma and epidermoid carcinoma metastases receiving plasmid encoding mouse IFN-α intramuscularly experienced a decrease in metastases as compared to control mice (page 105, line 18 - page 106, line 12).

The specification does not enable administering any non-infectious, non-integrating DNA as broadly claimed encoding IFN-α to treat cancer or metastases (claims 1, 66, 78). Applicants argue "plasmids" enable the breadth of non-infectious, non-integrating DNA. Applicants argument is not persuasive. The specification does not teach any other non-infectious, non-integrating DNA other than plasmids. The specification does not teach vectors other than plasmids having the same tissue targeting or express IFN-α to an amount that is therapeutic. Given the lack of predictability in the art regarding the combination of elements required to obtain

the desired effect taken with the teachings in the specification, it would require one of skill undue experimentation to determine a non-infectious, non-integrating DNA encoding IFN-α effective in treating cancer other than a plasmid.

The specification does not enable "active fragments" of IFN-α that would treat cancer or metastases, specifically to decrease tumor, increase survival or decrease metastases. Applicants argue the specification provides a definition of an active fragment of IFN-α as one that displays antiproliferative activity of the mature of full length cytokine (pg 32), assays for determining the antiproliferative activity of fragments and that making fragments of IFN-α are disclosed and routine. Applicants arguments are not persuasive. The art at the time of filing and the specification do not teach fragments of IFN-α capable of treating cancer or metastases. The specification does not provide an assay for determining the IFN-α fragments that are capable of treating cancer or metastases. In addition, a fragment having antiproliferative activity is not equivalent to a fragment capable of treating cancer. A fragment merely having "antiproliferative activity" in an assay may simply slow cell growth but not be capable of treating cancer in a patient. The teachings in the specification are simply a wish to know fragments of IFN-α having the ability to treat cancer in a patient. Given the teachings in the specification taken with what was known in the art at the time of filing, it would have required one of skill undue experimentation to determine fragments of IFN-α capable of treating cancer or metastases as claimed.

The rejection that the claims recite the DNA encoding IFN-α is operably linked to a promoter is withdrawn because the claims have been amended ("through operable association with one or more transcriptional control elements, wherein said one or more transcriptional control elements comprises a promoter").

The specification does not enable administering the composition to smooth muscle, myocardial tissue (claim 7), or any body cavity as broadly claimed (claim 66 and 78) for reasons of record. Applicant argument that Example 6 (pg 124, line 10) enables intraperitoneal injection of plasmid encoding IFN-α increased survival in mice having cancer is persuasive. Intraperitoneal injection does not enable the breadth of "body cavity" because "body cavity" encompasses numerous embodiments (stomach, mouth, nose, rectum, eye, etc.) which are not enabled. Losordo does not correlate to the elected invention because the plasmid encodes VEGF which has a materially distinct structure and function than IFN-α. The specification does not teach the effect of injecting IFN-α myocardially. In addition, Losordo was not available at the time of filing. The art at the time of filing and the specification does not teach administering plasmid encoding IFN-α to smooth muscle, the myocardium, or any body cavity other than the peritoneal cavity. The specification teaches intramuscular injection to striated muscle which results in systemic delivery of the plasmid. The specification does not correlate intramuscular injection to striated muscle to injecting smooth muscle, the myocardium, the peritoneal cavity or any other body cavity such that systemic delivery is obtained or cancer is treated. As such it cannot be determined whether injection into smooth muscle, the myocardium, the peritoneal cavity or any other body cavity

would also result in myocardial cells expressing IFN-α. The specification and the art at the time of filing do not teach the effect of expressing IFN-α in smooth muscle, the myocardium, the peritoneal cavity or any other body cavity. Given the lack of predictability in the art regarding the combination of elements required to obtain a desired effect using gene therapy taken with the teachings in the specification, the specification does not enable administering DNA encoding IFN-α to smooth muscle, the myocardium, the peritoneal cavity or any other body cavity as broadly claimed.

The specification does not enable obtaining cell-, tissue- or tumor-specific expression of IFN-α (claims 44 and 45) or "selectively transfecting malignant cells" (claim 78). Applicants argue pg 68-72 teach the construction of the DNA and pg 72, lines 2-5 suggest using "tissue-specific" promoters and enhancers. Applicants argument is not persuasive because the specification does not teach how specific the promoter or enhancer must be, because the specification does not teach any such promoters or enhancers and because the specification does not teach how to use such promoters or enhancers in combination with a vector encoding IFN-α to target a tissue of interest and treat cancer or malignant cells as claimed. The targeting of ascites (pg 111, line 24) is not specific as claimed delivery because ascites contains a number of different types of cells. Furthermore, pg 111, line 24, merely states tumor ascites is targeted. It does not state expression was exclusive in tumor ascites. Example 6 does not teach IFN-α was expressed exclusively in ovarian cancer cells. Applicants argue Example 7 shows how to "selectively transfect" malignant cells of interest. Applicants argument is not persuasive. While

the heading on page 126, line 10, states "selective transfection of tumor cells" the results (Tables 4-6) do not show tumor cells were targeted over non-tumor cells. In Table 6, pg 128, mesentery was not done ("nd") upon administering DNA+DMRIE:DOPE; however, mesentery showed expression in both other conditions. Therefore, administering DNA+DMRIE:DOPE would cause expression in tumor and mesentery. Thus, Example 7 does not teach selectively transfecting malignant cells as claimed. Overall, the specification teaches obtaining systemic delivery of IFN-α. The specification does not teach targeting IFN-α to a particular cell, tissue or tumor or the effect of such targeting. Given the lack of predictability in the art regarding targeting DNA to the desired tissue, taken with the teachings in the specification regarding the effect of targeting IFN-α to specific cells, tissues or tumors, the specification does not enable using cell-specific, tissue-specific or tumor-specific regulatory elements in the method as claimed.

The specification does not enable administering a vector encoding IFN-α as well as another cytokine (claim 42). Applicants argue pg 53, line 28, suggests making constructs encoding more than one cytokine and lists a number of cytokines (pg 54, lines 2-9). Applicants argue constructs encoding two cytokines were known in the art (Okada, 1997). Applicants arguments are not persuasive. A mere suggestion to add another cytokine to a vector along with a list of possible cytokines is not considered an enabling disclosure. What is required is a teaching as to the effect of the combination of the cytokine with IFN-α in treating cancer. The specification and the art at the time of filing did not teach how to use IFN-α and another cytokine to treat cancer. Within the realm of gene therapy, The specification and the art at the time of

filing did not teach how to use a vector encoding IFN-α and another cytokine. Given the lack of predictability in the art regarding the combination of elements required to obtain the desired effect using gene therapy taken with the lack of teachings in the specification regarding the effect of a vector encoding IFN-α and another cytokine, the specification does not enable using a vector encoding IFN-α and another cytokine as claimed.

2. Claims 1, 3-7, 9, 10, 15-18, 30-35, 38-50, 66, 67, 69-74, 77-81 and 83-86 remain rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

The rejection of claims 4, 39, 40 and 46 regarding the Markush group is withdrawn because double inclusion of an element by members of a Markush group is not sufficient basis for rejection of the claim (MPEP 2173.05(h), e.g. halogen and chloro-).

Claims 44 and 45 remain indefinite because the metes and bounds of "cell specific" and "tissue specific" cannot be determined for reasons of record. Applicants argue "cell specific" means expression is limited to the same type of cells (i.e. macrophages) or tissue. Applicants argument is not persuasive. The specification does not limit the meaning of "cell specific" or "tissue specific" to a particular type of cell or tissue. As such, it cannot be determined if the phrase encompasses limiting expression to one particular cell or one particular area of tissue. How specific is specific?

Claim 3 as newly amended is indefinite. It is unclear if the control elements include a promoter and a polyadenylation signal or if the promoter in claim 1 is replaced with the polyadenylation signal in claim 3.

Claim 43 as newly amended does not further limit claim 1 which already requires a promoter which is a "region regulating gene expression." As such, it is unclear if the cell specific or tissue specific regulating region in claims 44 and 45 is the promoter in claim 1 or some other region.

Claims 71-74 as newly amended are indefinite because "construct" or "polynucleotide construct" lacks antecedent basis in parent claim 66.

Claims 83-86 as newly amended are indefinite because "construct" or "polynucleotide construct" lacks antecedent basis in parent claim 78.

Claim Rejections - 35 USC § 102

Upon review of the application 09/196313, the rejection of claims 1-10, 15-18, 29-35, 38-50, 66-75 and 77-87 are rejected under 35 U.S.C. 102(b) as being anticipated by Horton (Feb. 1999, PNAS, Vol. 96, pages 1553-1558) is withdrawn.

The rejection of claims 66, 67, 69, 71, 78-80 and 83 under 35 U.S.C. 102(e) as being anticipated by Pestka (WO 97/00058, Jan. 3, 1997) is withdrawn because of the limitation "wherein said DNA is administered free from *ex vivo* cells".

Conclusion

Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL.** See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

No claim is allowed.

Inquiry concerning this communication or earlier communications from the examiner should be directed to Michael C. Wilson who can normally be reached on Monday through Friday from 9:00 am to 5:30 pm at (703) 305-0120.

Questions of formal matters can be directed to the patent analyst, Dianiece Jacobs, who can normally be reached on Monday through Friday from 9:00 am to 5:30 pm at (703) 305-3388.

Questions of a general nature relating to the status of this application should be directed to the Group receptionist whose telephone number is (703) 308-1235.

If attempts to reach the examiner, patent analyst or Group receptionist are unsuccessful, the examiner's supervisor, Deborah Reynolds, can be reached on (703) 305-4051.

The official fax number for this Group is (703) 308-4242.

Michael C. Wilson

MICHAEL C. WILSON
PATENT EXAMINER